HAZARDS OF COLD AIR

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Apart from the intrinsic hazards associated with cold weather athletic activities, winter sports enthusiasts face two potentially serious health hazards of the environment—frostbite and hypothermia. Injuries often develop without the athlete's awareness of warning signs or symptoms. Profound hypothermia can result ultimately in death, while serious frostbite can lead to the equally unfortunate end points of gross disfigurement and permanent disability. Prevention of such grave consequences rests in a thorough understanding of the nature and etiology of cold injuries, along with the application of judicious foresight and common sense. In the unfortunate circumstances where cold injury occurs despite all reasonable precautions, proper treatment can minimize the extent of the tragedy.

THERMAL BALANCE AND COLD INJURY

The cause of cold injury, reduced to its simplest description, is a failure to maintain thermal balance at physiologically acceptable levels. Failure at the whole body level results in hypothermia, whereas excessive regional heat loss leads to localized cold injury or frostbite.

The thermoregulatory system of the body uses both physical and chemical regulation, effected through vasomotor and metabolic control, respectively, to balance heat production against heat loss.

In a cold environment, the body must often contend with the conflicting requirements of maintaining the core temperature near 37°C, while preventing cold injury to the extremities. During vigorous physical activity in relatively mild cold, metabolic heat production may be more than adequate to sustain the temperature of both core and extremities. However, at times of lesser activity, or during severe environmental conditions, heat production may be insufficient, and the body "sacrifices the

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extremity to save the core". While this sounds like an active and deliberate thermoregulatory decision, it is a simple consequence of physiologic thermoregulation and the thermodynamics of heat transfer to and from the tissues.

The key to reducing heat loss is to increase insulation, precisely what the thermoregulatory system does through vasoconstriction. By restricting blood flow to the skin and extremities, the body allows these tissues to cool, thereby reducing heat transfer to the environment by all avenues (conduction, convection, radiation, and evaporation). The improvement in insulation thus obtained is, however, quite limited, and amounts to a total gain of less than 1 clo of insulation.* By comparison, extra layers of clothing can provide as much as 6 clo of insulation.

The amount of insulation available from clothing is limited by how much restriction in movement the activity will allow. On the other hand, the amount of insulation required to maintain thermal balance depends on the metabolic rate and the environmental conditions. These factors are often in conflict for various sporting activities; resolution of this conflict lies in layered clothing, which can be donned or doffed as the situation demands, and in the development of newer fabrics and clothing designs that offer superior insulation with less bulk and restriction of movement.

ENVIRONMENTAL FACTORS AND COLD INJURY

Environmental thermal stress is quantified by reference to the four meteorolgic factors of humidity, temperature, wind velocity, and radiation. Unitary indices based on weighted combinations of some of these factors have been developed for hot environments, but in the cold it is simpler to describe these factors and their relationships to cold injuries independently.

Atmospheric humidity is generally low during subfreezing weather, adding to the thermal stress of the cold environment. A minor but chronic effect of this lack of moisture is a drying of exposed skin. More acutely, heat and water are lost by evaporation of moisture from the respiratory tract. The fraction of total body heat loss attributed to respiration can become quite appreciable in the cold. Absolute respiratory heat loss increases with ventilation, but since ventilatory rate is closely coupled to metabolic rate, the fraction of heat lost by breathing varies little with activity at a given temperature. Evaporation of water during exercise is not compensated without a deliberate increase of fluid intake, and it contributes significantly to dehydration in the cold.

Accumulation of moisture within clothing is a serious concern during winter sports. Most insulating materials perform best when dry, and their thermal resistance rapidly diminishes with wetting. This wetting could arise from external sources such as rain or melting snow, and suitable waterproof outer garments should be used as appropriate. Wetting by sweat is an even more serious problem in cold climates, and physical activity should be adjusted if possible to avoid active sweating. If the metabolic rate is expected to increase substantially during a sporting activity, extra insulation should be removed to assist heat dissipation. If sweating is unavoidable, the wet garments should be removed or extra insulation added after the activity has been completed.

Temperature is the major environmental factor responsible for cold injury. It is not practicable to specify a critical temperature that delineates "safe" and "dangerous" environmental air temperatures regarding hypothermia. Avoidance of hypothermia depends upon body heat production offsetting environmental losses. There are, of course, practical limits to metabolic rate, body energy reserves, and clothing insulation. Ultimately, these factors determine tolerance and survival times under given conditions.

Since the freezing point of tissue is about −0.3 °C, it is virtually impossible to develop frostbite at ambient temperatures above this threshold. In practice, tissue supercools somewhat before ice nucleation sets in, and the threshold air temperature for frostbite may be as low as −10°C. The only exception to this critical temperature guideline is a spill of cold liquid on the skin. Fuels like gasoline, kerosene, and naphtha remain liquid to well below 0°C and can rapidly freeze any tissue they contact. Highly volatile liquids can even cause frostbite at temperatures above 0°C by rapid evaporation. Rubber gloves should be worn when handling such fluids in the cold.

Next to air temperature, wind is the second most important environmental factor responsible for cold injury. While everyone is familiar with the term "windchill factor", there are several misconceptions associated with this factor.

The first is that wind can cause an object to cool to a temperature below that of the ambient air. This notion is plainly contrary to the laws of heat transfer; it probably arises from the practice of expressing the effect of wind as an equivalent temperature, several degrees below the ambient air temperature. Wind can, however, increase the rate at which an object cools, causing frostbite to develop under conditions that would pose little or no threat in the absence of wind. For the winter athlete, wind can be created in relatively still air by body movement (for instance, in skiing or riding a snowmobile).

Another misconception is that the windchill equivalent temperature describes the degree of cooling perceived by the whole body. In reality, this relative measure of cooling applies only to an inanimate object initially at 33°C with no insulation. The only parts of the body that even approach this condition are the exposed regions of the face and bared extremities. No account is taken of metabolic rate or clothing. The same wind speed has a much greater cooling effect on nude or lightly clad individuals than it does on well-insultated persons (assuming the clothing has a windproof outer shell).

In spite of its shortcomings, the windchill factor is still of value as a guide to outdoor activities where

* One clo unit provides an insulation of 0.15°C/W/m². It is roughly equivalent, in practical terms, to a business suit.

exposed flesh is involved. Most windchill charts are divided into "safe" and "unsafe" zones, based upon estimated times for exposed flesh to freeze. Often, the exposed parts of the body dictate tolerance of a cold environment.

The last environmental factor to consider is radiation. All objects warmer than −273°C (absolute zero) can gain or lose heat by radiation. The net amount of heat exchanged between two objects depends upon the temperature difference between their surfaces. For well-insulated individuals in a cold environment, radiant heat losses are relatively minor, simply because the temperature difference between the surroundings and the clothing surface is small. Similarly, radiant heat loss from the body to the inner layer of clothing is minimal because of the small temperature difference between the two surfaces. Clearly, the use of "reflective" clothing over or under heavy insulation does little to reduce radiant heat loss. However, when a reflective layer is interposed between exposed warm flesh and a cold environment, or between a poorly insulated body and the environment, its effect becomes noticeable.

It is possible to gain considerable radiant heat from the sun even in the cold of winter. This raises the surface temperature of clothing, thereby reducing conductive heat loss from the body through the clothing. On sunny days, a reflective layer on the outside of clothing may be counterproductive.

OTHER EPIDEMIOLOGIC FACTORS

Age. Older persons are more likely to suffer chronic circulatory problems, making them more susceptible to cold injury. Young children, because of their small size, have a high surface area to volume ratio and lose heat at a faster rate than an adult.

Circulatory Problems. Any chronic condition (for example, Raynaud's syndrome) that interferes with the peripheral circulation predisposes to frostbite.

Fatigue. Mental fatigue and apathy can cause a neglect of warning signs, with failure to institute appropriate preventive measures. Physical fatigue compromises the defense against excessive heat loss, particularly if tissue reserves of glycogen are depleted.

Experience. Both frostbite and hypothermia can be prevented under most circumstances. When a mishap occurs (for example, getting lost, vehicle breakdown, climbing accident, or sudden weather change), persons experienced in cold survival are more likely to be dressed properly, to have extra food and clothing supplies, and to respond to the situation calmly and thoughtfully.

Race. Observations during World War II, during the Korean War, and in present-day Alaska confirm that the Negro is more susceptible to frostbite than the Caucasian.

Acclimation. Repeated exposures to cold stimuli can alter responses of the thermoregulatory system, increasing tolerance of severe conditions.

Concomitant Injury. Blood loss and shock reduce peripheral blood flow, predisposing to frostbite. After fracture, blood flow to the injured part may be limited due to severed blood vessels or constriction from bandages.

Previous Frostbite. Previous severe frostbite leaves the body more susceptible to cold injury for several years, perhaps due to slowly healing vascular and neurologic damage.

Drugs and Medication. Any drugs or medications that compromise vasomotor responses in the extremities should be used with extreme caution. Vasoconstrictors can lead to frostbite, while vasodilators predispose to hypothermia.

Alcohol. The effects of alcohol on heat exchange are controversial, but its known influence on judgment rule against its use in the cold. The percentage of frostbite and hypothermia victims who have consumed substantial amounts of alcohol prior to injury is alarmingly high.

FROSTBITE

Etiology. Frostbite occurs whenever tissue freezes, either intracellularly or extracellularly. During slow cooling, ice nucleation begins in the interstitial fluid, and cells respond to the increasing osmolality of their environment by dehydrating and shrinking. This loss of water reduces intracellular ice formation, but can cause osmotic injury to the cells. During rapid cooling, ice formation again begins extracellularly, but most cell types cannot dehydrate fast enough to avoid intracellular freezing. During rewarming, recrystallization causes the smaller ice crystals to grow disruptively, thereby inflicting physical damage upon cellular structures. Rapid rewarming generally minimizes such physical damage.

Regardless of the mechanism leading to injury, the target tissue ultimately responsible for the loss of flesh from frostbite appears to be the microcirculation. Time-lapse cinemicrography of frozen tissues has shown that the vascular system is functionally intact immediately after thawing, but that thrombi very quickly occlude the small capillaries. Electron microscopy of small blood vessels reveals extensive damage to endothelial cells. Presumably, thrombogenic agents released from these injured cells lead to clot formation with eventual total occlusion of the affected blood vessels.

Prevention. Prevention of frostbite is straightforward—avoid excessive cooling of body surface tissues. In particular, one must protect susceptible tissues such as the nose, cheeks, ears, fingers, and toes. Tissues most likely to suffer frostbite are those with a poor circulation, with a high surface area-to-volume ratio, at a distance from the body core, and with the greatest exposure to the elements. Insulation is the key to prophylaxis, but it must be used cautiously. Insulation that is excessive or constrictive can cut off the blood supply to an extremity. This is particularly true of hand and foot wear. If the source of heat is eliminated, the part cools toward ambient temperature like an inanimate object, the rate of cooling depending only upon the insulation between the tissue and the environment.

Marked blanching of the skin, numbness, loss of tactile sensation, or a sharp localized stinging (frostnip) are the first warning signs of frostbite. Rapid rewarming of the affected area with a warm hand will prevent the injury from becoming serious. However, it is then advisable to terminate the activity and seek shelter, since recurrence of the injury is highly probable.

Characterization. Frostbite injury can be graded, but the treatment for all categories is the same, since all frostbite looks identical both before and immediately after thawing. An assessment of severity can only be made in retrospect, leaving the clinician no choice but to treat all cases as severe injury.

First-degree frostbite is characterized by hyperemia and edema. After warming, the tissue becomes mottled, cyanotic, red, hot, and dry. There may be itching, burning, or a deep ache. Swelling and other symptoms may persist for several hours or even days, but the tissue usually heals with only minor peeling.

Second-degree frostbite is associated with vesicle formation, extending to the tips of the affected digits. The vesicles appear 6 to 12 hours after thawing and contain clear fluid. Only the superficial layers of the skin are affected, but after the blisters have healed, the tissue below may be very soft and sensitive to trauma.

Third-degree frostbite involves damage to the full skin thickness and to the tissue below. Vesicle formation is again present after thawing, but the blisters are filled with hemorrhagic fluid and rarely extend to the tips of the digits. Fleshy tissue loss is associated with this injury.

Fourth-degree frostbite involves injury to all tissues including bone. The development of post-thaw symptoms is slow. Vesicle formation is absent, and complete healing of the injury takes several weeks. If surgical amputation is not implemented, autoamputation of the mummified tissues occurs spontaneously.

Treatment. Provided the tissue is still frozen, the affected part is best thawed rapidly in a whirlpool bath controlled at 40°C. Water temperature should never exceed 42°C to avoid burning the already traumatized tissue. If the affected part is an ear or the nose, warm water can be poured over the injured area. Rapid warming may cause intense pain, requiring treatment with analgesics such as aspirin, Demerol, or even morphine. However, the benefit of reduced tissue loss far outweighs the drawbacks of the pain.

Warming should be terminated when thawing is complete, as indicated by the return of sensation (pain), color (red or purple), and softening of the affected area. The tissue should never be warmed above 37°C, and rapid warming should not be applied to thawed tissues. Rubbing with snow, massage, or the application of dry heat are to be avoided, since they increase tissue damage.

Severe frostbite should never be treated in the field, unless hospital facilities are hours out of reach. A frostbite victim can walk many miles on a frozen limb without incurring significant additional injury, but after thawing, such an individual becomes a stretcher case. Under no circumstances should thawing be initiated if there is a chance of refreezing, since this would greatly increase tissue loss.

Since tissue loss is mainly the result of microcirculatory failure, smoking and vasocontrictor drugs are contraindicated. On the other hand, anything that will promote vasodilation (such as warm fluids) is beneficial. Vasodilators, chemical or surgical sympathectomies, anticoagulants, and plasma expanders have all been used to treat frostbite, but the results are somewhat controversial. This is not surprising, since it is difficult to judge the effectiveness of treatment without knowing the extent of injury prior to treatment. Many of these treatments must also be started immediately after thawing in order to be effective.

Intra-arterial reserpine (0.5 mg) injected proximal to the injured extremity just after thawing and repeated at 2- to 3-day intervals for a week produces a temporary chemical sympathectomy with few systemic side effects. Fasciotomy is being used increasingly in cases where severe edema is impeding blood flow. Infusion of low-molecular-weight dextran, because of its risks, is reserved for cases in which tissue loss is highly probable. If used, treatment should consist of Dextran 40, 1.5 g/kg IV on the first day, followed by 0.75 g/kg IV daily for 5 days. The use of heparin as an anticoagulant has had mixed success and diathermy has not been adequately researched as a possible treatment modality in frostbite.

Follow-up care after the initial thawing is centered around open treatment under sterile conditions. Whirlpool treatment with 37°C water containing a disinfectant is given for 20 to 30 minutes two or three times a day. This promotes circulation, helps control infection, alleviates pain, and debrides the wound gently. During treatment, the patient is encouraged to flex the affected joints to avoid a flexion contracture.

If tissue loss appears certain, there may be a desire to amputate early to speed recovery. However, unless the line of demarcation between live and dead tissue is clear, or unless wet gangrene has set in, surgery should be delayed as late as possible.

HYPOTHERMIA

Etiology. Hypothermia has been defined clinically as the condition of a core (rectal) temperature of 34.4°C (94°F) or lower. It arises because the body loses more heat to the environment than it produces, and can occur at ambient temperatures well above freezing.

Acute hypothermia follows a rapid loss of body heat, as during sudden immersion in cold water. The thermoregulatory system has little time to respond before metabolic rate and heart rate become severely depressed. As a result, the body is "frozen" in a physiologic state similar to that prior to cooling.

Chronic hypothermia is the result of slow body cooling, as from prolonged exposure to cold wet conditions. In this instance, the thermoregulatory system has ample time to respond. Vasoconstriction shifts fluid from the periphery to the core, and the body responds with increased diuresis. Patients with chronic hypothermia are thus often volume depleted. The body responds to the heat loss with intense shivering, but as heart rate

and respiration decline, blood and oxygen supplies to the muscles decrease. Lactic acid and other metabolites accumulate in the tissues, leaving the patient acidotic. The resultant pH, electrolyte, and fluid volume disturbances become serious concerns during rewarming and treatment.

Prevention. Hypothermia is prevented by reducing heat loss to the environment. Appropriate clothing protects not only against cold, but also against wind and moisture. The usual recommendation is to wear several layers of loose clothing, topped with a windproof water-repellant shell. However, such clothing may be incompatible with an athletic activity, and many winter sports have evolved highly specialized styles of dress. In a sport such as cross-country ski racing, the clothing offers suitable protection only while the body is producing large amounts of metabolic heat, and additional clothing is required prior to and shortly after the activity has been completed. Conversely, a snowmobile suit offers excellent protection against the cold and wind while riding on the vehicle, but provides far too much insulation for the activity of building a snow shelter. Specialized clothing designed for specific activities can lead to cold injury if used inappropriately.

Often, the threat of hypothermia arises as a result of unforeseen circumstances such as getting lost, becoming injured, or falling in water. The first priority in such circumstances is to find shelter from the elements, particularly from the wind. Wet clothing should be substituted with dry, if possible. Conductive heat loss can be minimized by extra insulation, and wrapping the victim in a plastic sheet greatly reduces convective and evaporative heat losses. Additional external heat sources such as a fire, contact with a warm body (huddling), or warm fluids can further reduce cooling.

Prevention also involves recognizing the early symptoms of hypothermia. Initially, the body responds with an increase of shivering, but below a core temperature of 35°C, metabolism declines and shivering intensity wanes. With heat production now greatly reduced, the rate of body cooling increases substantially. Mental confusion, disorientation, and poor coordination are accompanied by mood changes such as depression and introversion. Simple tasks become difficult or impossible to perform. At a core temperature of about 32°C, cardiac arrhythmias may appear, and the victim may hallucinate and waver into semi-unconsciousness. Below 30°C, the victim is unconscious, with fixed, dilated pupils and no tendon reflexes.

As a rule of thumb, a person who is shivering violently but is reasonably alert may be becoming hypothermic; if shivering is weak or absent and mental confusion is profound, the patient is hypothermic and in serious danger.

Treatment. Profound hypothermia (unconsciousness; core temperature <30°C) should ideally be treated in a hospital with patient monitoring and life support equipment. Field treatment other than the prevention of additional heat loss is not recommended if hosptial facilities are nearby. If prompt evacuation is not possible, or if the victim is still conscious, field treatment should be initiated immediately. Under all circumstances, gentle handling is of the utmost importance in preventing cardiac fibrillation.

After rescue, the first step in treating hypothermia is to prevent additional heat loss (seek shelter, wrap in blankets and plastic sheet, etc.). A second priority is to establish a patent airway and encourage the patient to breathe. In the field, this may involve artificial respiration (mouth-to-mouth only) at 5 to 10 breaths/minute. In the hospital, endotracheal intubation may be preferrable, but care must be taken to avoid provoking fibrillation. Administration of 100 percent warm humidified oxygen under positive pressure is helpful, taking care not to overventilate and not to burn the air passages with steam. If cardiac monitoring is available, it should be utilized.

At this point, the decision has to be made whether to treat in the field or evacuate to a hospital. A cold body has a greatly reduced metabolic demand, and a short additional time in this condition adds little further injury. In contrast, if rewarming is begun in the field, it must be continued aggressively until the patient is rewarmed and can be moved to a hospital safely. If proper facilities can be reached within 30 minutes, one should opt for evacuation.

Rapid rewarming in a bath at 40°C is recommended if the core temperature is above 32°C. If the body is colder than this, the extremities should not be immersed, since this will induce extensive vasodilation. A rush of cold peripheral blood into the body core can lower the central temperature by as much as 2°C (the "afterdrop" phenomenon), inducing ventricular fibrillation if the heart cools to 28°C. In the field, rewarming by shivering may be all that is available. Administration of warm fluids or sitting near a fire assist heat gain, although some researchers caution that this could reduce the body's sensation of the cold and thus reduce shivering.

Chronic hypothermia presents special problems during rewarming because of the altered physiologic state. Although reestablishment of a normal body temperature is mandatory, treatment should also aim at correcting body fluid volumes, blood pH, and serum electrolytes, while avoiding cardiac problems. Intravenous fluids should be started as soon as possible (difficult with a vasoconstricted body), and a blood sample should be taken for analysis of serum electrolytes, blood gases, blood pH, and serum glucose. Blood gas readings must be corrected for the patient's temperature. When rewarming is begun, physiologic changes occur rapidly, and constant monitoring and correction of physiology are essential.

Hospital rewarming techniques can be divided into internal and external methods. Internal techniques include respiratory heating, peritoneal dialysis, mediastinal irrigation, extracorporeal circulation, hemodialysis, intragastric lavage, colonic lavage, and various forms of diathermy. Disadvantages of all these methods are that they require skilled medical teams, they take valuable time to implement, and they expose the patient to a risk of infection. Those who prefer internal rewarming cite the advantages as better control of warm-

ing rate, faster rewarming, and heating only of the core, thereby facilitating cardiac output while lessening the problems associated with afterdrop. External techniques include warm baths, radiant heat, electric blankets, hot water suits, heat packs, and showers. All methods of external rewarming accentuate the afterdrop and should only be used when the core temperature exceeds 32°C. They also suppress shivering to some degree, thereby compromising natural rewarming. The advantages are that they can be applied quickly, easily, and with little intrinsic risk to the patient. Some of them can, in fact, be implemented in the field.

During rewarming, respiratory support and correction of body fluids is continued. Temperature, blood pressure, blood gases, pH, and electrolytes are monitored frequently, while the ECG is watched constantly. Atrial arrhythmias and fibrillation occur often with hypothermia, but usually correct themselves with rewarming. Drugs to treat these symptoms are ineffective in a cold body. Ventricular arrhythmias and fibrillation are more serious, and should be treated. Premature ventricular contractions can be treated with lidocaine HCl (Xylocaine). Electrical defibrillation may be ineffective until core temperature has risen above 32°C, but it should be attempted at least once even at lower temperatures. Closed chest compression or a heart-lung machine may be used instead, if a normal rhythm is not restored.

After recovery, the patient should be monitored for several days. The most frequent complications are pneumonia, renal failure, pancreatitis, and frostbite. The thermoregulatory system may also be impaired for days or weeks, and cold environments should be avoided.

The most frequent question regarding field treatment of hypothermia concerns the use of cardiopulmonary resuscitation (CPR). Most CPR training is done with mechanical models, and most experience is gained with warm bodies. The response of a cold body to such manipulation is entirely different. In particular, the muscles of the chest are stiffer, and this will alter the "feel" of the treatment. The cold heart also does not respond in the way one would expect. Finally, the decision to attempt CPR may be based upon a failure to detect respiration or a palpable pulse; however, the hypothermic body has little requirement for oxygen, and hypothermic patients have been observed with a pulse rate as low as 2 beats/minute. It is thus recommended that CPR be attempted only when cardiac monitoring is available. In this way, the function of the heart can be clearly visualized, and the need for and effectiveness of CPR can be ascertained with little doubt.

Hypothermia victims frequently show no vital signs and are often mistaken for dead. However, low body temperature by itself is not a killer, since induced hypothermia to as low as 5°C is used frequently in surgery. It is essential, therefore, to try to resuscitate any cold body before it is pronounced as dead.